THE RECOVERY KERNEL

Substance-Specific Navigation Series

HOW TO RECOVER FROM NOVEL PSYCHOACTIVE SUBSTANCES

[Information provided here is for informational purposes only and does not constitute medical advice. Always consult with a qualified healthcare professional for any health concerns or before making any decisions related to your health or treatment.]

Recovery from novel psychoactive substances (NPS) can be challenging due to the unpredictable nature of these drugs and the potential for severe withdrawal symptoms. It's crucial to seek professional medical help immediately if you are experiencing withdrawal or adverse effects.

Here's a general overview of what recovery might involve:

*** **Medical Detoxification:** This is often the first step, involving supervised management of withdrawal symptoms under medical supervision. Medications may be**

used to alleviate discomfort and prevent complications. The length of detox varies depending on the substance(s) used and individual factors.

* **Assessment and Diagnosis:** A comprehensive assessment by a qualified healthcare professional is essential to determine the extent of the substance use, identify any co-occurring mental health conditions, and develop an individualized treatment plan.

* **Therapy and Counselling:** Various therapeutic approaches can be beneficial, including:
    * Cognitive Behavioral Therapy (CBT): Helps individuals identify and change negative thought patterns and behaviours associated with substance use.
    * Motivational Interviewing: Enhances motivation for change and helps individuals develop strategies to achieve their goals.
    * Group Therapy: Provides a supportive environment where individuals can share experiences, learn from others, and build coping skills.

* **Medication-Assisted Treatment (MAT):** In some cases, medications may be used to

reduce cravings, manage withdrawal symptoms, or address co-occurring mental health conditions. For example, in certain situations, medications like *Naloxone* might be necessary. Other medications that target specific receptors such as *COMT* and *DRD2* may also be considered under strict medical supervision. Genetic factors, such as variations in the *MTHFR* gene, can influence how individuals metabolize substances and may impact treatment decisions.

*   **Relapse Prevention:** Developing a relapse prevention plan is crucial for long-term recovery. This involves identifying triggers, developing coping mechanisms, and building a strong support system.

*   **Lifestyle Changes:** Adopting healthy lifestyle habits, such as regular exercise, a balanced diet, and adequate sleep, can significantly contribute to overall well-being and reduce the risk of relapse.

*   **Support Groups:** Connecting with others who have similar experiences can provide valuable emotional support and encouragement. *findahelpline.com* is a resource that may help you find local support groups.

* **Long-Term Follow-Up:** Ongoing monitoring and support are essential to maintain recovery and prevent relapse. This may involve regular check-ups with healthcare professionals, participation in therapy or support groups, and ongoing lifestyle changes.

Important Considerations:

* The specific treatment approach will vary depending on the individual's circumstances, including the substances used, the severity of the addiction, and any co-occurring medical or mental health conditions.
* Recovery is a process, not an event. It requires commitment, effort, and ongoing support.
* It's crucial to be honest with healthcare professionals about substance use history to ensure safe and effective treatment.
* Do not attempt to self-treat withdrawal symptoms. Medical supervision is essential to prevent complications.
* Medical help is not failure.

If you are concerned about your own or someone else's substance use, please seek professional medical advice. A *Recovery

Kernel* can be a valuable tool in managing the recovery process.

Synthetic Cathinones (Bath Salts) and Synthetic Cannabinoids (Spice/K2)

Using the Recovery Kernel Framework

Bath Salts · MDPV · Alpha-PVP (Flakka) · Mephedrone · Methylone / Spice · K2 · Black Mamba · Kush · Scooby Snax · Synthetic Weed

⚠ RESURSOS PARA REDUCCIÓN DE DAÑOS — NO ES UN CONSEJO MÉDICO | Crisis: llama al 000 o envía un mensaje de texto al 13 11 14.

SYNTHETIC CATHINONES: EXCITED DELIRIUM AND CARDIAC ARREST CAN OCCUR. CALL 000 FOR EXTREME AGITATION OR COLLAPSE.

SYNTHETIC CANNABINOIDS: THERE IS NO KNOWN REVERSAL AGENT. CALL 000 FOR SEIZURES, CARDIAC EVENTS, OR PSYCHOSIS.

These substances evade drug testing and scheduling. The specific compound may be unknown. The dose is always unknown.

How to Use This Guide

This guide covers two categories of novel psychoactive substances (NPS) that share a common origin — deliberate molecular engineering to evade drug scheduling — but have entirely different pharmacological mechanisms, dangers, and recovery architectures.

Category	Mechanism.	Primäre Gefahren	Family in recovery.
Synthetic cathinones (bath salts, MDPV, alpha-PVP, mephedrone)	Stimulant: inhibits the reuptake and promotes the release of dopamine, norepinephrine, and serotonin – similar to cocaine and MDMA, but with greater potency and a higher risk of causing psychosis.	Excited delirium, cardiac arrhythmia, psychosis, hyperthermia, death.	Stimulant recovery family (cocaine/meth guide framework) with critical amplifications.
Synthetic cannabinoids (Spice, K2, Black Mamba)	Full CB1 cannabinoid receptor agonist — significantly more	Psicosi, convulsão, evento cardíaco, lesão renal aguda,	Cannabis recovery program, with crucial

	potent than THC (partial agonist). Not cannabis.	sem agente de reversão.	enhancements for complete agonist receptor downregulation.

Find your category (Part A or Part B) and navigate directly. The integrated closing (Section 7) addresses both together.

PART A — SYNTHETIC CATHINONES

Bath Salts · MDPV · Alpha-PVP (Flakka) · Mephedrone · Methylone · Alpha-PHP · PV8

Kokainens arkitektur, MDMAs samhällsteknik, metamfetamins varaktighet – paketerat för att undvika klassificering.

Part A: A Note Before You Begin

Synthetic cathinones are beta-keto amphetamines — structurally derived from cathinone, the active compound in khat. They are the stimulant family's Novel Psychoactive Substance branch: designed to produce cocaine-like, MDMA-like, or methamphetamine-like effects through the same fundamental mechanism (monoamine reuptake inhibition and/or release), while being molecularly distinct enough to evade whatever scheduling is currently in effect.

The chemsex guide in this series addresses mephedrone and methylone in their most common clinical context — use in MSM sexual settings alongside crystal meth and GHB. That guide covers the chemsex-specific dimensions comprehensively. This guide addresses synthetic cathinones as a broader category: the MDPV and alpha-PVP (flakka) that devastated lower-income US communities with psychosis and excited delirium incidents from 2012 to 2016; the wider cathinone market that continues to evolve faster than scheduling can address it; and the recovery framework for people whose stimulant of choice happened to be a synthetic cathinone rather than cocaine or methamphetamine.

Il compendio applica il suo quadro sugli stimolanti: l'architettura della dopamina, la costituzione del gene Warrior COMT, la carenza del recettore DRD2 A1, l'architettura naturale dell'intensità del recupero. Ciò che questa guida aggiunge è cosa rende specificamente diverse le catinone sintetiche dagli stimolanti classici già trattati nella serie.

Section 0A — Immediate Safety: Synthetic Cathinones

⚠ EXCITATOIRE DELIRIUM ASSOCIÉ À L'MDPV ET À L'ALPHA-PVP EST UNE URGENCE MÉDICALE. UNE AGITATION EXTRÊME, UNE HYPERTHÉRMIE, UNE FORCE APPARENTEMENT SURHUMAINE ET UN COLLAPSE CARDIOVASCULAIRE PEUVENT SE PRODUIRE. APPELEZ LE 000. N'ESSAYEZ PAS DE CONTENIR PAR LA FORCE - CELA ACCENTUE LE RISQUE CARDIAQUE.

0A.1 — Excited Delirium: The MDPV/Alpha-PVP Emergency

Excited delirium (now often called Excited Delirium Syndrome or ExDS) is a medical emergency associated specifically with high-potency cathinone stimulants — MDPV and alpha-PVP most prominently. It does not occur with equivalent frequency from cocaine or methamphetamine. The syndrome reflects the unique combination of extreme dopamine dysregulation, severe hyperthermia, and the autonomic storm these compounds produce at their highest intensities.

Feature	What it looks like.	Okay, I understand. Just provide the text you want me to translate into Australian English, and I will follow all the rules you've outlined.
Intensa agitación y comportamiento agresivo.	La persona está en un estado de profundo terror y/o agresividad que no responde a la desescalada verbal. Es posible que no reconozca a personas conocidas. La fuerza y la insensibilidad al dolor parecen estar dramáticamente aumentadas.	Call 000 immediately. Do not attempt physical restraint — the catecholamine surge from forcible restraint compounds hyperthermia and cardiac risk. Maintain a calm voice and distance. Protect from self-harm and traffic.
Hyperthermia.	Core body temperature reaching 40–42°C (104–108°F). The skin is hot and flushed. The hyperthermia is the primary kill mechanism — the same mechanism as in crack cocaine and MDMA heat emergencies, but more severe and more common with MDPV/alpha-PVP.	Cool the person as much as possible — use cool water, a cool environment, and apply ice to the neck, armpits, and groin. This is life-saving while waiting for emergency services. Continue cooling until help arrives.
Cardiovascular collapse.	Dopo un periodo di agitazione estrema e ipertermia, può verificarsi un improvviso collasso cardiovascolare: fibrillazione ventricolare o altre aritmie. Questo può seguire sia la fase attiva di agitazione che il suo placamento.	CPR if collapse occurs. AED if available. Do not assume the person is "sleeping it off" if they suddenly become unresponsive after agitation. This is a recognised transition in excited delirium syndrome.
Extreme psychosis.	Paranoia, delusional thoughts, hearing or seeing things that aren't real, and losing touch with reality are all symptoms. These can be more severe and more common than with cocaine or methamphetamine, even when the stimulant doses are similar. MDPV and alpha-	Medical management — benzodiazepines for acute agitation (lorazepam or diazepam). Antipsychotic medication (haloperidol or droperidol) in emergency department settings. Tell EMS the specific substance: synthetic cathinone — MDPV or flakka — if

	PVP, in particular, have a high risk of causing psychosis.	known.

0A.2 — The Unknown Compound Problem

⚠ THE MOST DANGEROUS THING ABOUT SYNTHETIC CATHINONES IS THAT THE SPECIFIC COMPOUND IS UNKNOWN. 'BATH SALTS' IS A CATEGORY, NOT A SUBSTANCE. WHAT IS SOLD AS ONE CATHINONE MAY BE ANOTHER ENTIRELY, WITH DIFFERENT POTENCY, DURATION, AND TOXICITY.

Unlike cocaine (cocaine hydrochloride), methamphetamine (methamphetamine hydrochloride), or MDMA (3,4-methylenedioxymethamphetamine), 'bath salts' is a street category that may contain any of dozens of synthetic cathinone compounds, frequently mixed, with no labeling that reflects actual content. The dose is always unknown. The compound is always uncertain. This is the defining harm profile of this substance category and cannot be mitigated by any individual harm reduction practice beyond not using.

- Fentanyl test strips: although designed for opioids, carry them and test any powdered substance – fentanyl has been detected in cathinone supplies.
- Reagensia testen: Marquis-reagens zal verkleuren bij cathinonen, maar kan geen onderscheid maken tussen specifieke cathinonen of mengsels identificeren.
- Begin with very small quantities if testing is ongoing and the supply is uncertain.
- No uses solo — a state of agitated excitement and heart risks necessitate the presence of someone who can call emergency services.

Section 1A — What Synthetic Cathinones Did

Synthetic cathinones are a class of drugs that mimic the effects of stimulants like methamphetamine and cocaine. They can be found in powders, pills, capsules, or liquids, and are often sold under names like "bath salts" or "plant food."

Here's what synthetic cathinones do:

* **They affect the brain by increasing levels of dopamine and norepinephrine, which are neurotransmitters that play a role in mood, motivation, and attention.**
* **They can cause euphoria, increased energy, and alertness.**
* **They can also cause anxiety, paranoia, hallucinations, and seizures.**
* **In some cases, synthetic cathinones have been linked to death.**

If you are using synthetic cathinones, it is important to seek medical help immediately. Medical help is not failure. If you are having a bad reaction, call 000 or go to the nearest emergency room.

Here are some of the signs and symptoms of synthetic cathinone use:

* **Agitation**
* **Anxiety**
* **Confusion**
* **Hallucinations**
* **Paranoia**
* **Seizures**
* **High body temperature**
* **Rapid heart rate**
* **High blood pressure**
* **Nausea**
* **Vomiting**
* **Death**

If you are concerned about your synthetic cathinone use, please talk to a doctor or other healthcare professional. You can also find help online at findahelpline.com.

THE CENTURION — The Stimulant Architecture — Amplified and Destabilized.

Synthetic cathinones operate through the same fundamental mechanism as cocaine and methamphetamine: inhibition of monoamine reuptake transporters (DAT, SERT, NET) and/or reversal of these transporters to promote monoamine release. The specific pharmacological profile varies dramatically between compounds:

Compound.	Primær mekanisme.	N/A	Key distinction from classical equivalent.
MDPV (Methylenedioxypyrovalerone)	Potent DAT and NET reuptake inhibitor — no serotonin component.	Kokain — maar 10 keer krachtiger als een remmer van de dopamineheropname.	Pure dopamin/noradrenalin — no serotonergic dampening effect; directly associated with the most severe psychosis and excited delirium cases; extraordinarily

			compulsive due to a very short half-life (~30 minutes) and very high potency.
Alpha-PVP (α-Pyrrolidinopentiophenone) — 'Flakka'	DAT and NET reuptake inhibitor — similar to MDPV; slightly less potent.	MDPV — structurally similar, similar profile.	The compound most associated with the Florida 'flakka' epidemic (2014–2016). Highly lipophilic — rapid brain entry. Excited delirium, public psychosis incidents, 'superhuman strength' reports. Very high psychosis liability.
Mephedrone (4-methylmethcathinone, 4-MMC)	Releases AND inhibits reuptake of dopamine, serotonin, and norepinephrine — triple mechanism.	MDMA and cocaine combined — compendium lists it in the chemsex drug category.	Shorter-acting than MDMA; more compulsive due to shorter duration; less emphatic on the oxytocin-mediated connection quality; more stimulant-dominant. In the UK and Europe, most commonly encountered in club and chemsex contexts.
Methylone (beta-keto MDMA)	Triple reuptake inhibitor — similar to mephedrone but with a pharmacological profile closer to MDMA.	MDMA – but with a shorter duration, reduced empathogenic effects, and a stronger stimulant effect.	The most MDMA-like synthetic cathinone. Shorter duration drives more compulsive redosing than MDMA. Also appears in chemsex context.
Alpha-PHP, PV8, PV9, MPHP (pyrovalerones de geração mais avançada).	Inhibitors of DAT/NET with increasingly high potency – the "next generation" following the scheduling of MDPV and α-PVP.	MDPV — profilo simile, spesso più potente.	The constantly evolving 'next analog after scheduling' landscape. PV8 and PV9, in particular, are highly potent. They have long half-lives compared to alpha-PVP. Their prevalence is increasing in European new psychoactive substance (NPS) markets from 2020 onwards.

1A.2 — Why Synthetic Cathinones Cause More Psychosis Than Cocaine

THE CENTURION — The Pure Dopamine Problem: No Serotonin Brake.

The compendium's precision vocabulary section names cocaine's mechanism: 'triple reuptake inhibition.' Cocaine blocks DAT, SERT, and NET approximately equally. The serotonin component — the SERT blockade — is the mechanism that produces cocaine's social and somewhat empathogenic qualities and that may also provide a 'brake' on the pure dopaminergic drive.

MDPV και alpha-PVP έχουν ουσιαστικά καμία επιρροή στη σεροτονίνη. Είναι καθαρά φάρμακα ντοπαμίνης και νορεπινεφρίνης. Σε ισοδύναμη διεγερτική ένταση, η απουσία της σεροτονινικής ρύθμισης προκαλεί μια πιο ωμή, παρανοϊκή και ψυχωτική κατάσταση. Η έκρηξη ντοπαμίνης στο μεσελιμπικό σύστημα χωρίς την εξομαλύνουσα επίδραση της σεροτονίνης είναι η φαρμακολογική βάση της παρανοίας που προκαλείται από την κοκαΐνη, αλλά ενισχυμένη και πιο σταθερή.

For the person with a pre-existing psychosis vulnerability (COMT Met/Met, DRD2 A1 allele, personal or family history of schizophrenia or bipolar disorder) — MDPV and alpha-PVP represent a dramatically higher psychosis precipitation risk than equivalent dose cocaine. For people without those vulnerabilities, the psychosis risk at high doses is still substantially higher than for cocaine.

1A.3 — The Compulsive Redosing Architecture

Den mest klinisk vigtige adfærdsmæssige egenskab ved stærke syntetiske cathinoner er den tvangsmæssige gentagne dosering, som deres farmakokinetik kræver:

- MDPV half-life: approximately 30–60 minutes for the primary stimulant effect. The crash comes quickly. The craving for the next dose is immediate and complete.
- Alpha-PVP: has a similar short half-life. The "flakka binge" – days of continuous use with re-dosing every 30–60 minutes – is the typical heavy use pattern.
- The binge-to-crash cycle occurs over a shorter timeframe compared to crack cocaine. Consequently, the financial devastation happens more quickly. Neurological damage accumulates at a faster rate.
- The "fiend" pattern: the person cannot stop during a binge regardless of desire. This is not weak willpower — it is the pharmacokinetic trap of very high potency, very short duration, with each crash providing an overwhelming biological signal that the only available relief is the next dose.

Section 2A — Genomic Blueprint

Det genombibliotek för återhämtning från syntetiska katoniner är identiskt med det genombibliotek för återhämtning från stimulerande medel. Använd kokain/metamfetamin-guiden och dess genombiblioteksramverk, men med följande tolkningar som är specifika för katoniner:

Gene	Variant.	Cathinone-specific notes
COMT	Val158Met (Val/Val = Warrior)	Val/Val (Warrior) — the primary cathinone vulnerability profile, identical to cocaine. The fast dopamine clearance and intensity-seeking Warrior constitution was drawn to MDPV/alpha-PVP's extreme dopamine signal. Recovery architecture: same as cocaine/meth — the Warrior needs genuine high-intensity alternatives. The dual-COMT risk: Met/Met (Sage) combined with pure-dopamine cathinones (MDPV, alpha-PVP) creates the highest psychosis risk combination in the stimulant category.
DRD2 / ANKK1	Taq1A — A1 allele (reduced D2 density)	Reward Deficiency Syndrome underlying cathinone use — same as for cocaine, meth, crack. The A1 allele carrier's reward circuit has always been 'not enough.' MDPV's extraordinary dopamine potency was the first time the circuit ever fully fired. Recovery: Brain Stack (UMP + DHA + CDP-Choline) for receptor density upregulation. Most important structural supplement target.
DAT1 / SLC6A3	10-repeat (high DAT expression)	High DAT expression means rapid baseline dopamine clearance — the cathinone's DAT blockade produces a more dramatic relative effect because it is blocking a transporter that is clearing dopamine unusually fast. Also: ADHD association. A significant proportion of synthetic cathinone users have undiagnosed or undertreated ADHD that the stimulant effects were managing.
MTHFR	C677T, A1298C	Same methylation foundation as all stimulant recovery. 5-MTHF is often transformative for the mood deficit that cathinone stimulation was compensating for. MTHFR variants may have contributed to the baseline dopamine synthesis deficit that made the cathinone's dopamine flooding so compelling.
BDNF	Val66Met	Synthetic cathinone use — particularly the high-dose MDPV/alpha-PVP binge pattern — produces significant prefrontal BDNF suppression. Met carriers face slower cognitive recovery. Exercise (non-optional) is the BDNF driver. Semax peptide for accelerated prefrontal repair in heavy use histories.

Section 3A — Biological Reconstruction

THE CENTURION — The Stimulant Recovery Framework — Applied to the Most Potent Available Cathinones.

The biological reconstruction protocol is the cocaine and methamphetamine guides' framework applied directly. The cathinone-specific considerations are in the urgency and severity calibration — MDPV and alpha-PVP, because of their extraordinary potency and the severity of their binge pattern, produce more extreme dopamine depletion and more severe D2 receptor downregulation than equivalent-duration cocaine use. The recovery is the same framework, the timeline may be longer, and the underlying condition treatment (ADHD, depression) is more likely to be primary.

Supplement.	Dose.	Timing.	Cathinone-specific notes
5-MTHF + Methylcobalamin	5-MTHF 400–1000mcg; MethylB12 1000–2000mcg.	Morning.	Metilazione come base per la sintesi della dopamina (e serotonina per mefedrone/metilone). Per i portatori di varianti MTHFR, questo è spesso l'integratore più efficace da assumere inizialmente.
L-tyrosina eller Mucuna pruriens.	L-DOPA: Tyrosine 500–2000mg or Mucuna 100–300mg.	Good morning, skipping the protein today.	Precursor de dopamina. El objetivo principal para la reconstrucción de la dopamina. Para usuarios de mefedrona/metilonona: agregue 5-HTP (después de 3 a 5 días de abstinencia) junto con él, para abordar el aspecto serotoninérgico de la recuperación.
Brain Stack — Uridine + DHA + CDP-Choline.	UMP 250mg; DHA 1g; CDP-Choline 250mg.	Morning.	D1/D2 receptor density upregulation — the most important structural recovery target for the Reward Deficiency Syndrome profile. This is the compound that builds what MDPV was flooding. Takes weeks to months — begin on Day 1.
NAC (N-Acetylcystein).	600–1800mg dagligen, uppdelat.	Morning and evening.	Glutamate homeostasis — reduces cravings; an antioxidant to combat the severe oxidative stress associated with cathinone abuse. Several clinical trials have investigated its effectiveness in reducing stimulant cravings. Start on Day 1.
Magnesium	400–600mg	Evening.	Anxiety reduction; sleep support;

glycinate.	elemental		cofactor for dopamine synthesis. The anxiety associated with a cathinone "crash" – a pure dopamine crash without the moderating effect of serotonin – can be more severe than a cocaine crash for users of MDPV/alpha-PVP.
5-HTP (for mephedrone/methylone users only)	50–100mg	Evening, 3–5 days after cessation.	Rekonstruksi serotonin untuk komponen SERT dari katinhon (mefedron, metilon). Jangan tambahkan ke pemulihan MDPV/α-PVP tanpa bukti keterlibatan serotonin — ini adalah senyawa dopaminergik murni.

3A.2 — Management of Psychosis During Early Recovery

⚠ LA PSICOSIS INDUCIDA POR CATINONA PUEDE PERDURAR DÍAS O SEMANAS DESPUÉS DE LA INTERRUPCIÓN, A VECES MÁS QUE LA PSICOSIS INDUCIDA POR COCAÍNA O METAMFETAMINAS. SI LOS SÍNTOMAS PSICÓTICOS PERSISTEN MÁS DE 48 HORAS DESPUÉS DE LA ÚLTIMA TOMA: SE RECOMIENDA UNA EVALUACIÓN PSQUIÁTRICA.

- Akut psykos (under eller omedelbart efter användning): Benzodiazepiner är förstahandsval – diazepam 10 mg eller lorazepam 2 mg. Minskar agitation utan risken för QTc-förlängning som vissa antipsykotika medför.
- Persistent psychosis (beyond 48 hours): psychiatric evaluation. Short-course atypical antipsychotic (risperidone, quetiapine, olanzapine) while the dopamine system rebalances.
- Sleep as a primary psychosis intervention: psychosis severity in stimulant cessation correlates with sleep deprivation. Forced sleep opportunity (safe environment, dark, quiet) is the single most effective non-pharmacological intervention. Quetiapine at low dose (25–50mg) serves a dual purpose: sleep support and dopamine modulation.
- Monitor for persistent psychotic disorder: if psychosis does not resolve within 2–4 weeks of abstinence with adequate sleep, independent psychotic disorder (not substance-induced) must be considered. Psychiatric evaluation required.

3A.3 — The Craving Interrupt

THE CENTURION — El estimulante más rápido e intenso de su categoría, diseñado para un efecto MDPV de 30 minutos.

MDPV og alpha-PVP cravings are the fastest-converting cravings in the stimulant category. The combination of extreme potency, very short duration, and the pure-dopamine crash produces a craving signal that converts to use within minutes rather than the longer window of cocaine or meth cravings. The interrupt must work in that window.

1. IMMEDIATE INTENSE PHYSICAL INTERRUPT. Sprints, cold water, push-ups — something genuinely overwhelming that competes with the craving signal before it completes. This is the first 60 seconds.
2. MOVE PHYSICALLY AWAY FROM THE USE ENVIRONMENT AND SUPPLY. The most important single variable for the short-window cathinone craving.
3. CALL OR TEXT. Isolation is the most dangerous craving state for pure-dopamine cathinones. Connection is the counter-signal.
4. 4-7-8 BREATHING — EIGHT CYCLES. After the initial physical interrupt.
5. NAC e tirosina. Il precursore della dopamina e il composto per l'omeostasi del glutammato: supporto farmacologico per uno stato di desiderio farmacologico.
6. EXECUTE HIGHEST-INTENSITY NATURAL REWARD. From the Warrior natural architecture. The cathinone craving requires a real-intensity response — insufficient alternatives do not interrupt it.

3A.4 — Natural Architecture for Cathinone Recovery

The compendium's C-28 Warrior architecture applies directly. The person who was using MDPV or alpha-PVP for their extraordinary dopamine intensity was the Warrior seeking the loudest possible signal. The natural architecture for this recovery is identical to the crack cocaine guide's: vigorous physical training with a competitive element, mastery at the skill frontier, financial achievement, service. Everything real-intensity, genuinely demanding, genuinely rewarding.

For mephedrone and methylone users whose cathinone use was primarily in social or sexual contexts: the MDMA guide's natural porting framework (genuine connection, oxytocin-activating community, contemplative practice) supplements the stimulant recovery architecture.

PART B — SYNTHETIC CANNABINOIDS

Spice · K2 · Black Mamba · Kush · Scooby Snax · Legal Weed · JWH-018 · AB-FUBINACA · 5F-ADB

These are not cannabis. They are full CB1 agonists. The difference is clinically significant and potentially lethal.

Part B: A Note Before You Begin

Det viktigste å vite om syntetiske cannabinoider står på omslaget og gjentas her fordi det er den overordnede kliniske faktoren i hele denne veiledningen: dette er ikke cannabis. Det er ikke sterkere cannabis. Det er ikke det samme stoffet. De er fullstendige agonister ved CB1-cannabinoidreseptoren - en farmakologisk forskjell som gir dramatisk forskjellige og mye farligere effekter enn THC, som er en delvis agonist ved den samme reseptoren.

Spice, K2, Black Mamba, Kush, and their hundreds of compound variants have produced seizures, cardiac events, kidney failures, and deaths. They have been associated with psychosis rates far exceeding natural cannabis. They have been detected at autopsy in deaths that would be categorized as 'cannabis-related' if the specific compound analysis were not performed. They have devastated prison populations, homeless communities, and people in drug-tested employment who chose them specifically to avoid THC detection.

The cannabis guide in this series covers the endocannabinoid system, the CB1/CB2 receptor architecture, FAAH genetics, and the cannabis recovery framework. This guide addresses what is different — why the partial agonist distinction between THC and synthetic cannabinoids is not a matter of degree but of kind.

Section 0B — Immediate Safety: Synthetic Cannabinoids

⚠ NO REVERSAL AGENT EXISTS FOR SYNTHETIC CANNABINOID TOXICITY. THERE IS NO EQUIVALENT OF NALOXONE FOR CB1 RECEPTOR OVERDOSE. CALL 000 FOR ANY SIGNIFICANT ADVERSE EVENT.

Emergency.	Signs	Response
Psychosis / severe paranoia.	Meer dan gewoon cannabis, dit kan leiden tot ernstige problemen: intense paranoia,	Call 000. Ensure a safe environment. Use a calm voice and minimise stimulation.

	hallucinaties, opwinding en verlies van realiteitszin. Dit kan al binnen enkele minuten na gebruik optreden.	Administer a benzodiazepine (if available) for severe agitation – this helps reduce the anxiety component. Do not leave the person alone. Inform paramedics: synthetic cannabinoid – Spice/K2 – if known.
Krampe	Tonic-clonic seizures have been reported repeatedly in cases of synthetic cannabinoid toxicity. These seizures are not typically associated with natural cannabis, even at high doses. They can occur even during the first time using the substance.	Recovery position. Call 000. Time the seizure. Do not restrain. Do not put anything in the mouth. Paramedics are required – the seizure needs a medical assessment to rule out a metabolic cause and assess the risk of it happening again.
Cardiac events — tachyarrhythmia, MI.	Severe tachycardia (heart rate 150–200+), chest pain, irregular heartbeat. More common than with natural cannabis due to the more complete CB1 activation's cardiovascular effects and the direct cardiotoxicity of some synthetic compounds.	Call 000. The person should not exert themselves. Cardiac monitoring. Administer fluids for hypotension if present. Coronary artery spasm and myocardial infarction have been documented.
Acute kidney injury.	Specific to synthetic cannabinoids — not associated with natural cannabis. Flank pain, reduced urine output, vomiting. Associated with specific synthetic cannabinoid compounds in outbreak investigations.	Emergency evaluation. Acute kidney injury from synthetic cannabinoids requires intravenous fluid resuscitation and a nephrology assessment. Early presentation can be treated; delayed presentation risks permanent renal damage.
Hyperthermia.	Full CB1 agonism produces more complete hypothalamic dysregulation than THC partial agonism, including hyperthermia risk at high doses.	Cool the person. Call 000. Cooling is the treatment – follow the same protocol as for other heatstroke emergencies.

0B.2 — The Unknown Compound Problem — Even More Severe Than Bath Salts.

Synthetic cannabinoids face the same regulatory-evasion compound-change dynamic as synthetic cathinones — but with one additional complication. The potency variability between different synthetic cannabinoid compounds is even more extreme than between cathinones:

- First-generation compounds (JWH-018, JWH-073, 2012): moderate-high potency. Associated with psychosis and tachycardia but rarely life-threatening at typical doses.

- Third-generation compounds (AB-FUBINACA, 5F-ADB, MDMB-4en-PINACA, 2018 onwards): dramatically higher binding affinity and potency. What was a typical dose from older generation products is now a potentially lethal dose with newer compounds. Outbreaks of mass hospitalizations have occurred from contaminated herbal smoking product containing unexpectedly potent newer compounds.
- Concentration variability: Synthetic cannabinoids are sprayed onto inert herbal material. The spraying is not uniform—one section of the product may contain far more compound than an adjacent section. The first use from a bag may be mild; the next use from the same bag may cause seizure.
- No reliable testing: standard drug tests detect THC, not synthetic cannabinoids. This is a feature for users evading drug testing — and a hazard for anyone trying to understand what they have taken.

Section 1B — What Synthetic Cannabinoids Did

* Synthetic cannabinoids are drugs made to imitate the effects of marijuana. They are often sold online or in stores as "herbal incense," "potpourri," or "research chemicals." However, these products are not meant for human consumption and can be very dangerous.

* Synthetic cannabinoids affect the brain by binding to the same receptors as THC, the active ingredient in marijuana. However, synthetic cannabinoids are often much more potent than THC, which means they can cause stronger effects and a higher risk of side effects.

* The effects of synthetic cannabinoids can vary depending on the specific drug, the dose, and the individual taking it. Some common effects include:

    * Anxiety
    * Paranoia
    * Hallucinations
    * Seizures
    * Coma
    * Death

* Synthetic cannabinoids have been linked to a number of serious health problems, including:

- * Cardiovascular problems
- * Respiratory problems
- * Neurological problems
- * Mental health problems

* If you or someone you know is using synthetic cannabinoids, it is important to seek medical help immediately. Synthetic cannabinoid poisoning can be life-threatening.

[Image of a bag of "herbal incense" labeled "Not for Human Consumption"]

Important Note: The information provided in this section is not intended to be a substitute for professional medical advice. If you have any questions or concerns about synthetic cannabinoids, please talk to your doctor or other healthcare provider.

THE CENTURION — Full Agonist vs. Partial Agonist: The Distinction That Explains Everything.

La distinzione clinica tra cannabis naturale e cannabinoidi sintetici può essere ridotta a un unico fatto farmacologico: il THC è un agonista parziale del recettore CB1. I cannabinoidi sintetici sono agonisti completi del recettore CB1. Questa non è una differenza di quantità, ma una differenza di tipo.

CB1 receptor property.	THC (partial agonist)	Synthetic cannabinoids (full agonist).
Maximum mogelijk activatie (intrinsieke werkzaamheid).	THC can only produce a partial response at CB1 — even at 100% receptor occupancy, the response is limited by THC's partial agonism.	Full agonists produce the maximum possible CB1 response — the receptor fires at full capacity. The same receptor produces a qualitatively different magnitude of response.
Psychosis risk.	Cannabis psychosis risk is real, dose-dependent, and modulated by AKT1 and other genetic factors — but even at high doses, the partial agonism limits the maximal dopaminergic dysregulation.	Full CB1 agonism in the mesolimbic pathway produces more complete dopaminergic dysregulation. The psychosis risk is proportionally higher. Some synthetic cannabinoid compounds have produced psychosis at what would be 'first try' doses in drug-naive individuals.
Cardiovascular effects.	Mild to moderate tachycardia. Cardiovascular events are extremely rare at typical doses.	Severe tachycardia is common. Cardiac arrhythmia has been documented. Myocardial infarction has been documented in young people with no prior cardiac history.
Tolerance and withdrawal.	CB1 receptor downregulation with heavy use produces the cannabis withdrawal syndrome — milder than most substance withdrawals.	Downregulation of CB1 receptors is more pronounced with full agonists; consequently, the withdrawal syndrome tends to be more severe, manifesting as more intense insomnia, more intense anxiety, and more intense dysphoria. Seizures have been documented during synthetic cannabinoid withdrawal (which is not typical of cannabis withdrawal).
Toxicity to the kidneys.	Not associated with natural cannabis or THC.	Specific acute kidney injury pattern documented in multiple synthetic cannabinoid outbreak investigations. Mechanism unclear — may involve direct nephrotoxicity of specific compounds rather than CB1 activation per se.

1B.2 — The Drug Testing Evasion Population.

THE CENTURION – The substance people used because they had to pass a test – and why this shapes recovery.

A defining feature of synthetic cannabinoid use is the population context: unlike most substances in this series, synthetic cannabinoids are often chosen not because they are preferred but because they evade drug testing for THC. The populations most affected:

- Populations in prisons and jails: Cannabis-derived THC can be detected in urine drug tests administered in correctional settings. Synthetic cannabinoids were for years undetectable on standard panels. The result: an extraordinarily high prevalence of synthetic cannabinoid use in correctional settings, associated with outbreaks of psychosis, seizures, and cardiac events that have turned these facilities into sites of medical emergencies.
- Personal militar: motivos semelhantes para evitar testes de drogas. O uso de canabinoides sintéticos em contextos militares resultou em eventos adversos documentados e gerou uma resposta significativa nas políticas militares.
- Drug-tested employment: People in safety-sensitive positions who use cannabis recreationally may switch to synthetic cannabinoids to pass workplace drug tests.
- Probation and parole: drug testing compliance requirements create the same evasion motive as incarceration.

The implication for recovery: the person who used synthetic cannabinoids primarily to pass a drug test has a different recovery architecture than the person who used them as a preferred substance. For the testing-evasion user, the recovery may involve addressing the underlying cannabis use disorder AND the institutional context that created the evasion motive — potentially including advocacy for more accurate drug testing that detects both THC and synthetic cannabinoids.

1B.3 — The Endocannabinoid System Under Full Agonist Attack.

The compendium's endocannabinoid entry in the C-28 natural addiction porting table: 'Cannabis → Calm, creativity, escape boredom → Endocannabinoid system, mixed COMT → Creative pursuits, nature, structured calm.' This framework applies to synthetic cannabinoids, but with greater severity at every point.

The endocannabinoid system's primary functions — stress response modulation, pain modulation, appetite, mood, sleep, and the 'runner's high' endocannabinoid anandamide effect — are all mediated through CB1 and CB2 receptors. Full CB1 agonism by synthetic cannabinoids produces more complete receptor downregulation with heavy use, more severe disruption of all these functions, and a withdrawal syndrome that reflects the more complete receptor suppression.

Recovery requires rebuilding natural CB1 activation through the same mechanisms the compendium identifies: creative pursuits that produce anandamide through flow

states, nature and awe (documented anandamide activators), exercise (the 'runner's high' is endocannabinoid-mediated), and the structured calm of contemplative practice.

Section 2B — Genomic Blueprint

Gene	What it controls.	Synthetic cannabinoid recovery relevance.
CNR1	CB1 cannabinoid receptor gene — the primary target of synthetic cannabinoids.	CNR1 variants affect baseline CB1 receptor density and sensitivity. High-expression CNR1 variants may experience more potent synthetic cannabinoid effects and more severe withdrawal from full agonist use. CNR1 variants are also associated with cannabis use disorder — and the synthetic cannabinoid user with CNR1 high-risk variants may have had an underlying predisposition to cannabinoid system dysregulation before any substance use.
AKT1	Serine/ threonine kinase — modulates dopamine signalling in response to cannabinoid CB1 receptor activation in the striatum.	AKT1 rs2494732 T/T genotype is strongly associated with cannabis psychosis risk — and applies to synthetic cannabinoid psychosis risk with even greater force, because the full CB1 agonism produces more complete AKT1-pathway dopamine dysregulation than THC. The cannabis guide identifies AKT1 as the primary psychosis risk gene; it is equally relevant here and the threshold for psychosis is lower.
FAAH	Fatty acid amide hydrolase – enzyme that breaks down anandamide (the endogenous cannabinoid).	FAAH C385A variant (AA genotype) produces lower FAAH activity — slower anandamide breakdown — meaning higher baseline anandamide tone. People with AA genotype have lower anxiety and less exogenous cannabinoid seeking at baseline. They may have used synthetic cannabinoids in a context where the endocannabinoid system was already functioning adequately, making the recovery easier once the exogenous compound is removed. CA/CC genotype: lower baseline anandamide → more endocannabinoid deficit → more severe synthetic cannabinoid withdrawal → more difficult recovery.
COMT	Clearance de la dopamine : risque de psychose par le biais de la dimension dopaminergique	Met/Met (Sage) + AKT1 T/T is the highest synthetic cannabinoid psychosis risk combination. The Sage nervous system's already-elevated dopamine and catecholamine tone, combined with AKT1-mediated dopamine dysregulation from full CB1 agonism, creates a specific neurobiological configuration for psychosis precipitation. This combination should be considered a

	de l'activation du CB1.	near-absolute contraindication to synthetic cannabinoid use.
MTHFR	Methylation — affects the endocannabinoid system indirectly through homocysteine and methylation-dependent processes.	MTHFR variants affect homocysteine levels, which influence the endocannabinoid system through several indirect pathways. The primary recovery relevance: 5-MTHF supplementation supports the anxiety, depression, and cognitive repair that synthetic cannabinoid withdrawal and heavy use produce through the methylation-dependent neurotransmitter synthesis dimension.

Section 3B — Biological Reconstruction: Synthetic Cannabinoid Recovery.

THE CENTURION — More Severe Than Cannabis Withdrawal — Because the Receptor Downregulation Is More Complete.

3B.1 — The Withdrawal Timeline

Timeline.	Biology.	Experience.	Management.
Hours 12–24	CB1 receptor upregulation beginning; endocannabinoid system attempting to compensate for withdrawal of full agonist.	Anxiety, insomnia, cravings, irritability — similar to cannabis withdrawal but more intense.	Magnesium glycinate, L-theanine. Low-sensory environment. CBD (if available and legal — see 3B.3) may reduce withdrawal severity through partial CB1/CB2 modulation.
Days 1–4 (peak)	Maximum CB1 receptor upregulation; endocannabinoid system at maximum deficit; sympathetic nervous system activation.	Severe insomnia (the most prominent symptom), profuse sweating, nausea and vomiting, tremor, anxiety, restlessness. In some cases: seizure (medical emergency — call 000). More severe than cannabis withdrawal in most measures.	Risco de convulsões: se houver sintomas de abstinência graves, a supervisão médica é apropriada. Benzodiazepinas controlam tanto a ansiedade quanto o risco de convulsões. Hidroxizina para dormir. Suplementos que auxiliam no funcionamento do GABA (magnésio, L-teanina, taurina).

Weeks 1–4	CB1 receptor density recovering; sleep architecture slowly normalizing; anxiety beginning to resolve.	Miglioramento con variabilità. PAWS: ansia episodica, ricomparsa dell'insonnia, forti voglie — scatenate da stimoli ambientali e stress.	Full supplement stack. Exercise daily from Week 1. The cannabis recovery sonic protocol applies: delta binaural beats for sleep; 80–100 BPM for daytime; fresh genres not associated with using.
Months 1–6	Substantial CB1 receptor recovery; natural endocannabinoid function gradually returning; BDNF supporting neuroplastic repair.	Progressive recovery. Natural calm, creativity, and stress management capacity gradually returning through endocannabinoid system restoration and the natural activation practices.	Construir la arquitectura endocannabinoide natural (Sección 3B.4). Tratamiento para el TDAH si estaba presente un TDAH subyacente. Tratamiento para la depresión/ansiedad.

3B.2 — The Biological Reconstruction Stack

Supplement	Dose.	Timing.	Synthetic cannabinoid-specific notes
Magnesium glycinate.	400–600mg elemental	Morning and evening.	GABA-A receptor support and NMDA modulation for the anxious hyperexcitability of synthetic cannabinoid withdrawal. The most immediately effective supplement for the anxiety and insomnia of withdrawal. Begin Day 1.
L-Theanine	200–400mg	Morning and evening.	Modulation of GABA and glutamate; anxiolytic effect without daytime sedation; sleep support in the evening. Directly relevant to the CB1 withdrawal anxiety mechanism.
5-MTHF + Methylcobalamin	5-MTHF 400mcg; B12 1000mcg	Morning.	Metilazione come base per la sintesi dei neurotrasmettitori e il recupero in tutti i sistemi compromessi dalla downregolazione del recettore CB1, in particolare nelle dimensioni della serotonina e della dopamina dell'interazione tra sistema endocannabinoide e monoaminergico.

NAC	600–1200mg twice daily.	Morning and evening with food.	Glutamate homeostasis; anti-neuroinflammatory; craving reduction. The CB1 receptor-glutamate interaction means NAC's glutamate-normalizing effect is specifically relevant for synthetic cannabinoid withdrawal.
Omega-3 DHA + EPA	2–4g daily	Breakfast.	Sistema endocannabinoide: supporto per le membrane. I recettori CB1 sono incorporati nelle membrane fosfolipidiche neuronali; il ruolo del DHA nel supportare le membrane favorisce direttamente il recupero della densità dei recettori. Effetto anti-infiammatorio nei confronti dell'infiammazione neurologica causata dall'uso di cannabinoidi sintetici.
Taurine	1000–2000mg	Morning.	GABA-mimetic; cardiovascular support — relevant given synthetic cannabinoids' documented cardiac toxicity; anxiety reduction. The same rationale as taurine in energy drinks and stimulants, but via the GABAergic rather than adrenergic mechanism.
Melatonin (low dose)	0.5–1mg sublingual.	30–60 minutes before bed, at a consistent time.	L'insonnia causata dall'astinenza dai cannabinoidi sintetici è il sintomo più frequentemente segnalato e più angosciante. L'assunzione regolare di basse dosi di melatonina fornisce un segnale circadiano senza creare dipendenza farmacologica. Combinata con magnesio glicinato e glicina (3g), questa protocolla per il sonno affronta il sintomo più debilitante dell'astinenza.

3B.3 — CBD: The Legitimate Endocannabinoid Harm Reduction Tool.

THE CENTURION — Cannabidiol is not a synthetic cannabinoid; it is a completely different compound.

CBD (cannabidiol) is frequently confused with THC and with synthetic cannabinoids because it comes from the same plant family. It is pharmacologically distinct from both:

- CBD does not significantly activate CB1 or CB2 receptors. It does not produce the psychoactive effects of THC or the severe effects of synthetic cannabinoids.
- CBD has negative allosteric modulation at CB1 receptors — it slightly reduces CB1 receptor activation from other cannabinoids. It may reduce the severity of both cannabis and synthetic cannabinoid withdrawal through this mechanism.
- CBD has documented anti-anxiety and antipsychotic properties. It has been used in research specifically for cannabis use disorder, and there is emerging evidence for its use in managing withdrawal from synthetic cannabinoids.
- If legal where the person is located: CBD oil (not a hemp-based CBD product from unknown sources but pharmaceutical-grade CBD where available, or high-quality documented-purity CBD oil) may support synthetic cannabinoid withdrawal management alongside the full stack. Dose range: 150–600mg daily has been used in cannabinoid withdrawal research.
- CBD does not cure synthetic cannabinoid withdrawal. It may reduce severity. It is not a substitute for the medical management of severe withdrawal (which requires benzodiazepines and possibly antipsychotics).

3B.4 — Natural Endocannabinoid Architecture

The compendium's C-28 framework for cannabis: 'Creative pursuits, nature, structured calm.' For synthetic cannabinoid recovery — where the CB1 system has been more completely suppressed — these natural activators are both more difficult to access in early recovery (because CB1 is more thoroughly downregulated) and more important for the long-term restoration of natural endocannabinoid tone.

- Exercise — specifically sustained aerobic exercise: the 'runner's high' is mediated by the endocannabinoid system, specifically anandamide release, not endorphins as historically believed. Sustained aerobic exercise (45+ minutes) produces the most reliable natural anandamide release available. This is pharmacologically specific to endocannabinoid recovery.
- Exposition à la nature et émerveillement : les environnements naturels sont reconnus comme des activateurs de l'anandamide par les mêmes mécanismes que l'émerveillement, à savoir l'activation du système parasympathique et la diminution de l'attention portée au soi. Pour la récupération après une consommation de cannabinoïdes synthétiques, le temps passé dans la nature n'est pas seulement agréable ; il s'agit d'une activation biologiquement pertinente du système endocannabinoïde.
- Creative flow states: the flow state (challenge at the skill frontier) activates anandamide alongside dopamine, serotonin, norepinephrine, and endorphins. For the person whose synthetic cannabinoid use was primarily about escape from boredom or stress: creative mastery is the structural answer.

- Meditatie en contemplatieve praktijken: de vermindering van de normale, op zichzelf gerichte activiteit van het default mode network is geassocieerd met de afgifte van anandamide - hetzelfde mechanisme als het endocannabinoïde component van 'flow'- en 'verbazing'-ervaringen. Meditators die langere tijd mediteren, vertonen een verhoogd endocannabinoidniveau.
- Lion's mane mushroom (500–1000mg daily): The compendium explicitly names lion's mane as a BDNF/NGF stimulator with good clinical evidence. Neurotrophin support for the CB1 receptor system recovery and the cognitive repair that synthetic cannabinoid use impairs.

3B.5 — The Craving Interrupt for Synthetic Cannabinoids.

Synthetic cannabinoid cravings share characteristics with cannabis cravings (environmental cue-triggered, driven by anxiety and stress) but with the additional complication of the testing-evasion motive — for the person in a drug-tested environment, the craving for a 'clean' option may be specifically directed toward synthetic cannabinoids even in recovery.

7. Is this an endocannabinoid deficit craving (anxiety, restlessness, need for calm), a stress-triggered cue, or a testing-evasion rational ('I need to pass my test')? Each requires a different response.
8. 4-7-8 BREATHING — SIX CYCLES. The parasympathetic activation of the 4-7-8 pattern directly activates the same calming pathways that the CB1 receptor was mediating — it is the closest available non-pharmacological substitute for the anxiolytic effect.
9. EXPOSITION À LA NATURE OU ACTIVITÉ PHYSIQUE INTENSE. L'alternative naturelle la plus spécifique sur le plan pharmacologique à l'activation du récepteur CB1 est la libération d'anandamide, que ce soit par l'une ou l'autre de ces voies.
10. FOR TESTING-EVASION CRAVINGS: Address the testing context. If on probation or parole — discuss the testing panel with your legal team. Many panels have been updated to detect specific synthetic cannabinoids. The 'safe' testing evasion option may no longer be either safe or evading. The clinical framework for this: the harms of synthetic cannabinoid use far exceed the harms of testing positive for cannabis in most contexts.
11. CBD (si applicabile): se il CBD è disponibile, l'effetto ansiolitico acuto può ridurre la voglia nello stesso intervallo di tempo in cui NAC e magnesio vengono assorbiti.
12. CONTACT. The isolation that synthetic cannabinoid use enables (no smell, no paraphernalia, no detectable metabolites) also allows it to continue without social accountability. Connection is the counter-substance.

Section 7 — The Integrated View: Novel Psychoactive Substances and the Regulatory Chase.

THE CENTURION — The molecule changed. The biology didn't. The recovery framework was always the same.

Synthetic cathinones and synthetic cannabinoids exist because chemists can modify molecules faster than regulators can schedule them. The compounds keep changing. The pharmacological families stay the same. MDPV was scheduled; alpha-PVP emerged. Alpha-PVP was scheduled; MDPHP emerged. JWH-018 was scheduled; AB-FUBINACA emerged. AB-FUBINACA was scheduled; 5F-ADB emerged. Each new compound was more potent, less studied, and more dangerous than its predecessor.

The people caught up in this regulatory crackdown weren't seeking novelty for its own sake. They were choosing based on availability, affordability, or to avoid detection in drug tests. The populations—people in prisons using synthetic cannabinoids because THC testing fails them, people in low-income communities using MDPV as a cheap cocaine substitute, and people in chemsex contexts where mephedrone and methylone were the accessible options—were not the intended targets of the regulatory systems that constantly changed which compounds were prohibited. They were the unintended victims.

Recovery from synthetic cathinones is stimulant recovery. The dopamine architecture, the Warrior intensity needs, the natural high-intensity framework — these apply to the compounds identically to cocaine and methamphetamine. The cathinone-specific additions are the unknown-compound problem, the excited delirium risk that exceeds the classical stimulants, and the pure-dopamine psychosis liability of MDPV and alpha-PVP.

Recovery from synthetic cannabinoids is cannabinoid recovery — but calibrated for the more complete CB1 receptor downregulation of full agonism. The endocannabinoid natural architecture (exercise-anandamide, nature, creative flow, contemplative practice) is the same framework. The withdrawal severity, the seizure risk, the kidney toxicity, and the psychosis liability are specific to the full agonist pharmacology and require the amplifications this guide provides.

For both categories: the Recovery Kernel's foundation applies without modification. The substance masking analysis — what was the substance providing? what does that signal point to? — produces clear answers. Extreme stimulant intensity (Warrior constitution, RDS). Calm, creativity, and stress escape (endocannabinoid deficit, CNR1). The answers determine the natural architecture. The natural architecture is the recovery.

La molecola è cambiata. La ripresa è sempre stata la stessa.

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NPS harm reduction: talktofrank.com | Drug policy information: drugpolicy.org | Crisis: 988 | SAMHSA: 1-800-662-4357

Refer to the Recovery Kernel Complete Centurion Compendium for all referenced protocols.

Crisis: call or text 988 | Emergency: 911 | SAMHSA: 1-800-662-4357

How to Recover from Novel Psychoactive Substances Using the Recovery Kernel | Substance-Specific Navigation Series.